Successful Management of Aluminium Phosphide Poisoning Resulting in Cardiac Arrest

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Aluminum phosphide has high toxicity when it is ingested, and in case of contact with moisture, phosphine gas is released. Aluminum phosphide poisoning causes metabolic acidosis, arrhythmia, acute respiratory distress syndrome and shock, and there is no specific antidote. A 17-year-old male patient was referred to our hospital because of aluminum phosphide poisoning with 1500 mg of aluminum phosphide tablets. The patient's consciousness was clear but he was somnolent. Vital parameters were as follows: blood pressure: 85/56 mmHg, pulse: 88 beats/min, SpO₂: 94%, temperature: 36.4°C. Because of hypotension, noradrenaline and dopamine infusions were started. The patient was intubated because of respiratory distress and loss of consciousness. Severe metabolic acidosis was determined in the arterial blood gas, and metabolic acidosis was corrected by sodium bicarbonate treatment. In addition to supportive therapy of the poisoning, haemodialysis was performed. Cardiac arrest occurred during follow-ups in the intensive care unit, and sinus rhythm was achieved after 10 min of cardiopulmonary resuscitation. The patient was discharged after three sessions of haemodialysis on the ninth day. As a result, haemodialysis contributed to symptomatic treatment of aluminum phosphide poisoning in this case report.

Keywords: Aluminum phosphide, poisoning, hemodialysis

Introduction

Aluminium phosphide is a disinfectant used against pests and insects that damage stored grain; phosphine is released when aluminium phosphide comes in contact with moisture. Being exposed to phosphine at a dose of 1400 mg m⁻³ for 30 min can be fatal because it is a toxic gas. Aluminium phosphide use is limited in northern Europe (1). Most deaths due to cardiovascular toxicity usually occur within the first 12–24 h. In toxicity, the heart, lung, kidney and gastrointestinal tract are the most affected. The effects of phosphine on the heart are multifactorial, and the most common cause of mortality is cardiogenic shock (2).

Phosphine inhibits cytochrome c oxidase. Similar to ischemia, the inhibition of mitochondrial respiration results in myocardial energy depletion. It also causes the formation of reactive oxygen species due to lipid peroxidation. These effects may lead to changes in cardiac transmembrane action potential (3). In this case report, we aim to provide the successful management of aluminium phosphide poisoning treated by haemodialysis therapy despite the development of cardiac arrest.

Case Presentation

A 17-year-old male patient was referred to our hospital because of an attempt to commit suicide with the intake of a 1500 mg of aluminium phosphide tablet. In his physical examination performed in our hospital, the Glasgow coma score was 13, and he was drowsy 6 h after the intake of the tablet. His vital parameters were as follows: TA, 85/56 mm Hg; KTA, 88/min; SpO₂: 94% and temperature, 36.4°C. He was admitted to the anaesthesia and resuscitation intensive care unit (ICU) and was started on infusion with 200 mL h⁻¹ of isotonic saline, 10 mcg kg⁻¹ min⁻¹ of dopamine and 0.1 mcg kg⁻¹ min⁻¹ of steradin. Gastric lavage was performed. Applied laboratory tests are shown in Table 1. The patient was intubated and connected to a mechanical ventilator on the 30th hour of his admission to ICU due to loss of consciousness and development of respiratory distress. Sodium bicarbonate treatment was started when severe metabolic acidosis was detected in his arterial blood gas analysis (pH: 7.06, PO₂ 167 mmHg, PCO₂ 17 mmHg, HCO₃: 4.8 mEq/L, BE: -25.5 and SpO₂: 99%). Due to intoxication, haemodialysis was performed when his metabolic acidosis improved. In his follow-up, the patient developed

cardiac arrest, and sinus rhythm was achieved after applying cardiopulmonary resuscitation for 10 min. Haemodialysis was repeated on the second and third days of his admission with dopamine and steradin support. On the fifth day of hospitalization, dopamine and steradin infusion was stopped, and he was extubated on the seventh day. After being observed extubated in ICU for two days, he was discharged.

Discussion

When aluminium phosphide is mixed with aqueous solutions, phosphine is released, and it is quickly absorbed from the intestines and lungs. The fatal dose of phosphine is 150–500 mg for a 70-kg person. Aluminium phosphide has high toxicity when taken. The toxicity of phosphine is associated with inhibitory metabolism enzymes such as cytochrome c oxidase and oxygen free radicals (4). It is toxic to the lungs, heart and blood vessels and causes pulmonary oedema arrhythmia and shock.

The most common clinical implications of poisoning are nausea, vomiting and dopamine-persistent hypotension (5). Hypermagnesaemia is also seen due to damage in the myocardium and liver (6). Majority of deaths are seen within the first 24 h. The patient who was admitted to our hospital 6 h after aluminium phosphide intake had a low blood pressure, and his clinical findings were consistent with the clinical picture of aluminium phosphide poisoning. Although vasopressor

therapy was started and haemodialysis was performed, the patient developed cardiac arrest. We believe that cardiac arrest was caused by the negative effects of phosphine on the cardiac conduction system and myocardial metabolism. The increase in CK-MB levels also confirms myocardial damage. The early haemodialysis that we started prevented the progression of cellular damage by allowing the removal of free oxygen radicals. After haemodialysis was started in the following days, an improvement in laboratory results was observed.

Singh et al. (5) reported that between 1989 and 1995, 115 of 195 patients who had been accepted to have aluminium phosphide poisoning died. They also observed that while hypotension and metabolic acidosis were more common among those who died, nausea and vomiting were more common among the survivors (5). Singh et al. (7) stated that all 121 patients in their study had nausea and vomiting. In addition to nausea and vomiting, shortness of breath, palpitation, cyanosis, hypotension and shock were observed in the patients (7).

There is a significant relationship between heart and ECG findings in aluminium phosphide poisoning, and troponin-T positivity may result in mortality. Soltaninejad et al. (8) reported in their study that in aluminium phosphide poisoning, ST-segment elevation was observed on ECGs of patients due to the effects of phosphine (8).

Table 1. Results of biochemical parameters studied in our hospital

	Emergency room (day 1)	ICU (day 1)	After dialysis (day 2)	After cardiac arrest (day 2)	Before discharge (day 9)
Glucose (mg dL ⁻¹)	123	163	134	350*	184
BUN (mg dL ⁻¹)	13.1	34.4*	23	28.3	19.8
Creatinine (mg dL ⁻¹)	0.98	2.1*	0.74	1.01	0.48
SGOT (u L ⁻¹)	15	93	454	506*	41
SGPT (u L ⁻¹)	11	47	285	346	134
Uric acid (mg dL ⁻¹)	-	14.04*	7.3	10.84	4.27
CK (u L ⁻¹)	-	299	2028*	-	108
CK-MB (u L ⁻¹)	-	85*	-	-	57
Sodium (mmol L ⁻¹)	142	140	137	138	138
Potassium (mmol L ⁻¹)	4.1	6.20*	3.8	4	3.7
Chlorine (mmol L ⁻¹)	106.8	106.9*	103	99	99
Calcium (mg dL ⁻¹)	-	7.8	7.1	6.9	7.3
Magnesium (mg dL ⁻¹)	-	2.48	1.99	2.26	1.78
Phosphorus (mg dL ⁻¹)	-	10.81*	3.82	7.31	3.1
Total bilirubin (mg dL ⁻¹)	-	2.37	5.32*	2.95	0.92
BUN: blood urea nitrogen; SGOT: aspartate aminotransferase; SGPT: alanine aminotransferase; CK: creatine kinase; CM-MB: creatine kinase-MB. * The highest result in the patient's laboratory follow-up.					

Personnel may also be affected in aluminium phosphide poisoning as a result of the release of phosphine. Among workers dealing with the spraying of stored agricultural products, minor symptoms such as coughing, shortness of breath, chest pressure, headache, dizziness, drowsiness, lethargy, anorexia and epigastric pain have been reported. Therefore, to prevent exposure, air-tight containers and chemical protective suits can be used.

Treatment is symptomatic without any antidote. Besides symptomatic treatment, dialysis in the early stage is also recommended after aluminium phosphide intake. In addition to other resuscitation efforts, Nasa et al. (9) managed the survival of two patients with aluminium phosphide poisoning by renal replacement therapy that they started in the early period. In addition to supportive therapy, glutathione and magnesium, which were reported as having N-acetylcysteine and antioxidant properties, are also recommended. In rats exposed to aluminium phosphide, while N-acetylcysteine decreased oxidative damage in the myocardium, it increased survival time. By changing the myositis metabolism and reducing the oxygen consumption, trimetazidine may also have a potential role in cardioprotection (10).

Conclusion

We believe that in preventing high mortality due to aluminium phosphide poisoning, implementing haemodialysis at an early stage and taking precautions against metabolic acidosis, arrhythmia, ARDS and shock are necessary.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

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